Periodontitis: A risk factor for pre-eclampsia

Sir

Periodontitis is a destructive inflammatory disease of the supporting tissues of the teeth and is caused either by specific microorganisms or by a group of specific microorganisms, resulting in progressive destruction of periodontal ligament and alveolar bone with periodontal pocket formation, gingival recession, or both.[1] Periodontal diseases are recognized as infectious processes that require bacterial presence and a host response and are further affected and modified by other local, environmental, and genetic factors. Inflamed periodontal tissues produce significant amounts of pro-inflammatory cytokines, mainly interleukin 1-beta (IL-1β), IL-6, prostaglandin E2, and tumor necrosis factor-alpha (TNF-α), which may have systemic effects on the host. Periodontitis initiates systemic inflammation and can be monitored by inflammatory markers like C-reactive protein or fibrinogen levels.[2] The host responds to the periodontal infections with an array of events involving both innate and adaptive immunity.[3] Periodontitis has been proposed as having an etiological or modulating role in cardiovascular and cerebrovascular diseases, diabetes, respiratory disease, and adverse pregnancy outcome.[4] Recently, pre-eclampsia has been proposed to be a syndrome caused by an excessive systemic inflammatory response to pregnancy. It is a rapidly progressive condition observed during pregnancy, characterized by hypertension and the presence of protein in the urine. At least 3–5% of pregnancies are affected, resulting in high morbidity and mortality around the world.[5] The pathophysiologic abnormalities of pre-eclampsia are numerous. Some of the reported abnormalities include placental ischemia, generalized vasospasm, abnormal hemostasis with activation of the coagulation system, vascular endothelial dysfunction, abnormal nitric oxide and lipid metabolism, leukocyte activation, and changes in various cytokines as well as in insulin resistance.[6] The clinical course of severe pre-eclampsia may be characterized by progressive deterioration in both maternal and fetal conditions. Because these pregnancies have been associated with increased rates of maternal morbidity and mortality and with significant risks for the fetus (growth restriction, hypoxemia, and death), there is universal agreement that all such patients should deliver if the disease develops after 34 weeks' gestation.[7] Periodontal disease may burden pregnant women systemically with endotoxin, inflammatory cytokines, and oxidative stressors at the maternal–fetal interface; thus, it may be a vascular stressor that plays a role in the development of pre-eclampsia in pregnant women.[8] This host response to a long-term exposure of periodontal pathogens may provoke systemic maternal and placental pro-inflammatory endothelial activation and dysfunction, which represent a significant risk factor for diseases of vascular origin, such as pre-eclampsia.[9] Future research should focus on large scale longitudinal studies as well as interventional studies to better prove a causal relationship and determine if periodontal treatment or prevention reduces the risk for pre-eclampsia.

Rajiv Saini

Department of Periodontology and Oral Implantology, Rural Dental College, Loni, Maharashtra, India

Address for correspondence

Dr. Rajiv Saini
Department of Periodontology and Oral Implantology,
Rural Dental College, Loni, Rahata,
Ahmednagar, Maharashtra – 413 736, India.
E-mail: drperiodontist@yahoo.co.in

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